Intensive Care Med
https://doi.org/10.1007/s00134-025-07975-1

WHAT'S NEW IN INTENSIVE CARE

Central venous pressure: current uses and prospects for an old parameter

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Despite being a pivotal parameter in hemodynamic management, central venous pressure (CVP) fell out of favor when several meta-analyses demonstrated its inability to predict fluid responsiveness (FR) [1, 2]. CVP is measured in the superior vena cava via a jugular or subclavian catheter and serves as a surrogate for right atrial pressure (RAP). Zeroing to the midpoint of the right atrium ('phlebostatic axis') is crucial since even small differences may induce high variability in measured values [3]. Measurement is ideally performed at end-expiration at minimal intrathoracic pressure, at the base of the c-wave. The CVP displayed by the monitor provides a reasonable estimation in spontaneously breathing patients (SBP), with irregular respiratory patterns [4]. Beyond the value, the appearance of the waveform can offer diagnostic insights into various pathologies. There may be three distinct uses:

CVP as the backpressure of venous return

At equilibrium, venous return (VR) (volume of blood returning to the right atrium per minute) equals cardiac output (CO). According to Guyton's model, VR is proportional to the pressure gradient for VR (Eq. 1) divided by venous resistance:

$$\text{VR gradient} = \text{Mean systemic filling pressure (Pms)} - \text{RAP}$$

where RAP = CVP.

Pms, a theoretical pressure at the venular level, increases with stressed blood volume (the portion of blood maintaining venous transmural pressure above zero) and decreases with vascular compliance. VR can be increased by raising Pms through fluid infusion (via expansion of total blood volume) or enhancing venous tone (via venoconstrictors or sympathetic stimulation by reducing venular capacitance). The heart functions as a pump that creates a pressure gradient between Pms and CVP, with CO dependent on VR. CVP reflects the heart's pumping efficiency (greater efficiency leads to lower CVP), which cannot significantly raise Pms due to arterial resistance. CVP ranges from 0 to 6 mmHg (rarely exceeding 3–4 mmHg in SBP), while Pms varies between 7 and 10 mmHg. This narrow VR gradient means that even small increases in CVP can significantly reduce VR and CO (Fig. 1).

CVP as the downstream pressure for organs

Blood flow in most of the organs is tightly related to mean perfusion pressure (MPP) calculated as the difference between systemic mean arterial pressure (MAP) and CVP. The regulation of perfusion pressure is a key determinant of organ function and may be compromised in critical illness [5]. It is particularly true in heart failure, where its association with liver dysfunction and impaired microcirculatory flow is well documented [6]. In sepsis, an increased CVP—rather than MAP alone—is linked to venous congestion and a reduction in MPP, which in turn is associated with a higher risk of acute kidney injury [7]. Although there is evidence supporting the association between high CVP and organ dysfunction, establishing causality is less straight forward [7–9].

CVP as a reflection of right ventricular (RV) filling pressure

Under physiological circumstances, transmural CVP (tCVP) is a good estimation of the transmural RV diastolic pressure. During mechanical ventilation (MV), the effect of transmitted intrathoracic pressure on the CVP should be considered. This may be estimated by applying an analogous method to the one proposed by Teboul

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et al. for estimating transpulmonary artery wedge pressure [10]:

$$\text{tCVP}\left(\text{mmHg}\right) = \text{intraluminal CVP} - [((\text{PEEP}) * (\Delta\text{CVP}))/((\text{Pplat} - \text{PEEP}))],$$

where ΔCVP is the difference between peak-inspiratory and end-expiratory CVP, Pplat is the end-inspiratory airway pressure, and PEEP is the positive end-expiratory pressure.

As a surrogate of RV filling pressure, high tCVP indicates either supraphysiological RV overdistension or reduced diastolic compliance. The consequence would be a decrease in VR and a potentially overdistension upstream (venous congestion). In critical care, MV commonly elevates CVP by increasing RV afterload and should not be automatically interpreted as myocardial dysfunction or hypervolemia. In a recent study, among 518 MV patients, those with RV dysfunction (RVD) had significantly higher CVP [11]. CVP showed good accuracy in identifying a combined endpoint RVD and venous congestion (AUC = 0.839), with an optimal cutoff of 10 mmHg but was less reliable for general RVD [12]. A large gray zone (9–12 mmHg) limits its predictive value to diagnose RVD but may make it useful to rule out RV failure ((i.e., RV dilatation with congestion) [12].

Potential applications for CVP in hemodynamic management

Given its physiological basis and reasoning by a so-called gray zone approach, extreme CVP values may help define preload limits (approximately 1/3 patients receiving MV) [13]. Very low CVP values may indicate low cardiac preload, whereas high values suggest that fluid administration could be harmful. However, this has not been demonstrated in clinical settings, as absolute CVP values do not discriminate between FR and non-responders, and changes in CVP during passive leg raising (PLR) or heart–lung interactions are unreliable for this purpose [14]. However, Lakhal et al. suggested that an increase in CVP of more than 2 mmHg during PLR may enhance the predictive value of pulse pressure variation observed during the same maneuver [15].

As a downstream pressure for organs, CVP plays a key role in venous return and high CVP is associated to worse outcomes [9]. This raises the question of whether specific CVP thresholds should prompt interventions to reduce it, aiming for the lowest feasible CVP while maintaining adequate CO [7]. Feasibility has been explored in specific settings. In ARDS patients, a restrictive strategy guided by CVP after stabilization was linked to shorter MV duration [16]. In clinical practice, the decision to continue fluid administration in a patient showing signs of congestion despite preload dependency warrants early re-evaluation to avoid potential harm [17].

Finally, CVP may assess venous congestion in the setting of RV failure, a condition often marked by preload unresponsiveness and independently associated with mortality when combined with RVD [18, 19] (see Fig. 2).

Despite the justified abandonment of CVP to predict FR, it remains a valuable bedside tool. We should now aim to better define the scope of its interpretation. It is high time to move beyond the CVP value and consider its dynamic variations as a mean of monitoring the complex interplay between VR, RV and lungs.

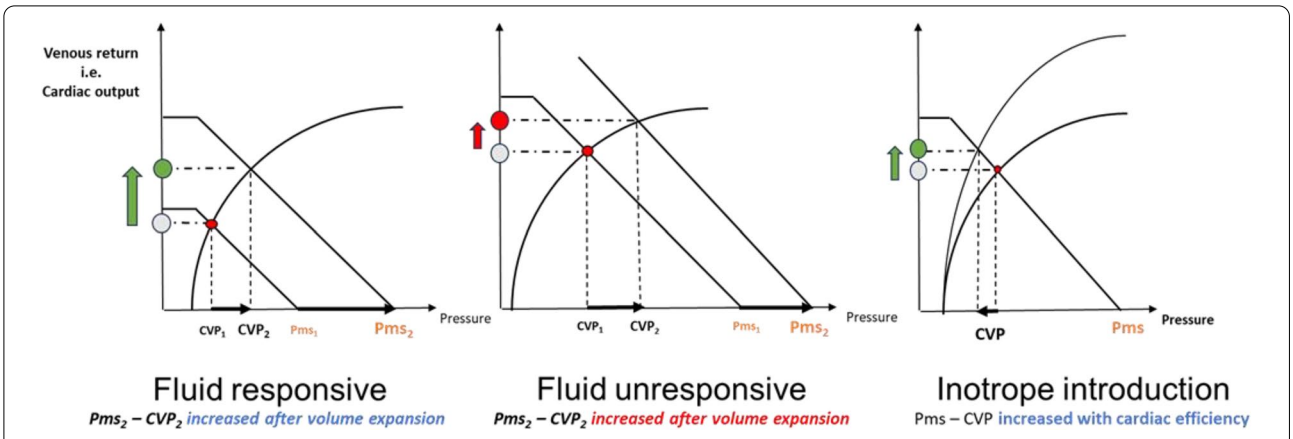

Fig. 1 Relationship between venous return and Frank–Starling curves illustrating effects on central venous pressure (CVP) and mean systemic filling pressure (Pms)

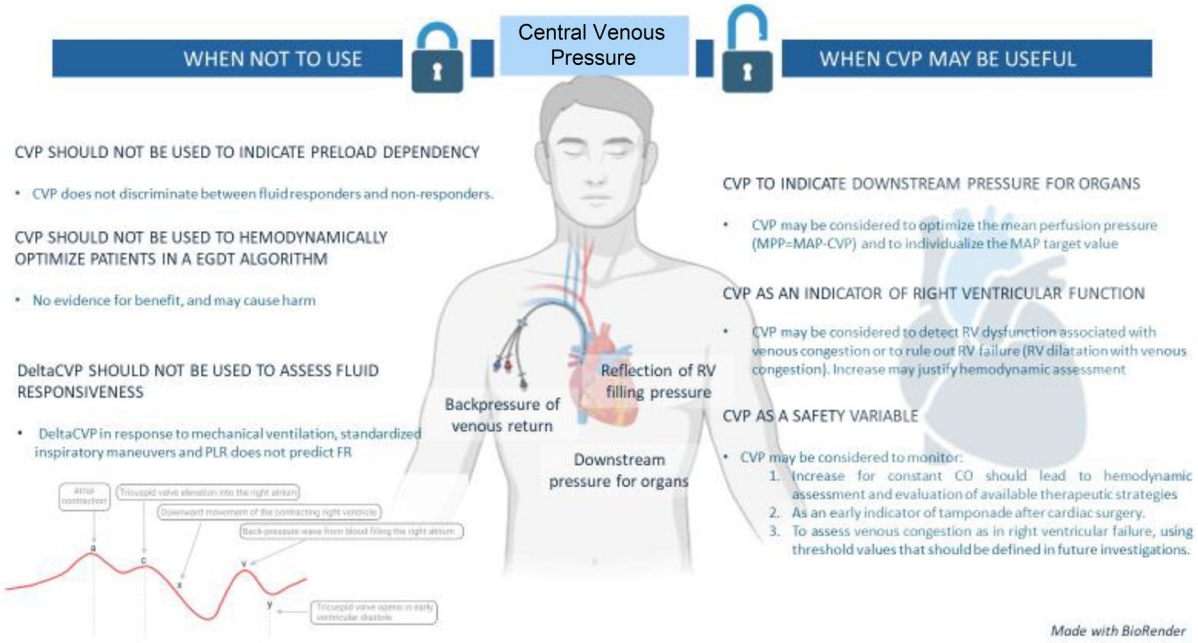
WHEN NOT TO USE
Central Venous Pressure
WHEN CVP MAY BE USEFUL
CVP SHOULD NOT BE USED TO INDICATE PRELOAD DEPENDENCY
• CVP does not discriminate between fluid responders and non-responders.
CVP SHOULD NOT BE USED TO HEMODYNAMICALLY OPTIMIZE PATIENTS IN A EGDT ALGORITHM
• No evidence for benefit, and may cause harm
DeltaCVP SHOULD NOT BE USED TO ASSESS FLUID RESPONSIVENESS
• DeltaCVP in response to mechanical ventilation, standardized inspiratory maneuvers and PLR does not predict FR
CVP TO INDICATE DOWNSTREAM PRESSURE FOR ORGANS
• CVP may be considered to optimize the mean perfusion pressure (MPP=MAP-CVP) and to individualize the MAP target value
CVP AS AN INDICATOR OF RIGHT VENTRICULAR FUNCTION
• CVP may be considered to detect RV dysfunction associated with venous congestion or to rule out RV failure (RV dilatation with venous congestion). Increase may justify hemodynamic assessment
CVP AS A SAFETY VARIABLE
• CVP may be considered to monitor:
1. Increase for constant CO should lead to hemodynamic assessment and evaluation of available therapeutic strategies
2. As an early indicator of tamponade after cardiac surgery.
3. To assess venous congestion as in right ventricular failure, using threshold values that should be defined in future investigations.
Backpressure of venous return
Reflection of RV filling pressure
Downstream pressure for organs
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Fig. 2 When is central venous pressure helpful? *CVP* central venous pressure, *EGDT* early goal directed therapy, *PLR* passive leg raising, *FR* fluid responsiveness, *MPP* mean perfusion pressure, *MAP* mean arterial pressure, *RV* right ventricular, *CO* cardiac output

Abbreviations
CO: Cardiac output; CVP: Central venous pressure; FR: Fluid responsiveness; MAP: Mean arterial pressure; MV: Mechanical ventilation; PLR: Passive leg raising; PEEP: Positive end-expiratory pressure; RAP: Right atrial pressure; RV: Right ventricular; SBP: Spontaneous breathing patients; VR: Venous return.

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Acknowledgements
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Author contributions
MC: literature search, screening for material, and drafting manuscript for intellectual content. MSC: instigator, literature search, proof-reading manuscript for intellectual content, and checking references. OH: instigator, proof-reading manuscript for intellectual content, and checking references.

Funding
Not applicable.

Declarations

Conflicts of interest
MSC is a Section Editor for Intensive Care Medicine journal.

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Received: 13 April 2025 Accepted: 26 May 2025
Published online: 25 June 2025

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